

REMARKS

Applicants respectfully request reconsideration of the present application in view of the foregoing amendments and in view of the reasons which follow.

I. Introduction

Upon entry of the above amendment, claims 1-12 are pending. Applicants have revised claims 1, 2 and 7-12 to address the examiner's concerns raised in relation to indefiniteness rejection. Applicants have replaced formula I on page 3 of the specification with new formula that shows the linking group X. According to the examiner's request, applicants have submitted herewith a separate sheet of abstract. Applicants also have amended the specification to incorporate a Brief Description of the Drawings.

The amendments made herein are not intended to further limit the claims or acquiesce to the propriety of the examiner's position in any rejection. The amendments made herein are introduced to advance the case towards allowance.

II. Rejection under 35 USC § 112, Second Paragraph

The examiner has rejected claims 1, 2 and 7-13 as allegedly indefinite for various reasons. Applicants have addressed each rejection below.

The examiner has objected to the recitation of "prophylactic or therapeutic inhibition" in claims 1 and 13. Without acquiescing to the propriety of the examiner's rejection, applicants have obviated the rejection by canceling claim 13 and revising claim 1 to be directed to "a method of inhibiting the activity of a toxic material or substance in a human or non-human animal patient." Support of the amendment can be found throughout the specification, for example, at page 9, lines 4-15. As described in the specification, the term "inhibition" is used in the instant case to encompass both prophylactic and therapeutic treatments.

With respect to the rejections of claims 2 and 7-11, applicants have revised these claims to attend to the examiner's various concerns, which renders these rejections moot.

In view of the forgoing amendments, applicants respectfully request withdrawal of all the indefiniteness rejections.

III. Rejection under 35 USC § 102(b)

The examiner has rejected claims 1-13 as allegedly anticipated by Matthews *et al.* (WO 95/34595). Applicants respectfully traverse this rejection.

At the outset, applicants wish to draw the examiner's attention to revised claim 1, which is directed to a method of inhibiting the activity of a toxic material or substance in a patient.

Matthews *et al.* allegedly discloses the use of the recited compounds as an antiviral agent. Yet the reference is silent about an inhibitory effect of the recited compounds on the activity of a toxic material or substance. Thus, the examiner seems to rationalize the anticipation rejection by equating the use of the recited compound for an antiviral activity with the use for the inhibition of the activity of a toxic material/substance.

Contrary to the examiner's understanding, however, one of ordinary skill in the art would easily understand that an "antiviral activity" refers to the inhibition of the replication of the virus, not to the inhibition of any toxic material/substance originated from virus.

It is generally understood that, once viral infection takes place, the genetic material of a virus is transferred to the host cell and the virus can 'take over' by incorporating its DNA into the DNA of hosts. The infected cell essentially becomes a factory for manufacturing virus. Almost all viral infections result in the death of the host, but in rare cases viruses leave their host cells alive. When this happens the cells are normally damaged beyond repair. With each successive transmission between hosts, a virus is able to replicate itself thousands of times.

Thus, an "antiviral" agent is generally defined as a substance, drug, or process that destroys a virus or suppresses its replication and, hence, inhibits its capability to multiply and reproduce. SEE DORLAND'S ILLUSTRATED MEDICAL DICTIONARY, W.B.

Saunders Company (1988), and Med Terms. com — Medical Dictionary — <http://www.medterms.com/Script/Main/art.asp?li=MNI&ArticleKey=8147>).

The appended excerpt from FIELDS VIROLOGY, 3rd edition (pages 431-433) further supports the proposition that inhibition of viral replication is what is understood to be the action of an antiviral agent. More specifically, the publication states that

...The major drawback in developing antivirals has been an inability to distinguish viral replicative mechanisms from host processes. Nevertheless, progress has been made over the past two decades in discovering molecules necessary for virus replication, characterizing them mechanistically, and in developing antiviral agents to inhibit them...Antiviral agents have been discovered and characterized in increasingly sophisticated ways. The most straightforward approach involves screening compounds for their ability to interfere with virus replication in cell culture...

In contrast, a “toxin” is generally understood to be “a colloidal poisonous substance that is a specific product of the metabolic activities of a living organism and is usually very unstable, notably toxic protein when introduced into the tissues, and typically capable of inducing antibody formation.” MERRIAM-WEBSTER MEDICAL DICTIONARY. Thus, a toxin is not an infectious agent, like a virus. As a result, an antiviral agent such as a HIV protease inhibitor or a reverse transcriptase inhibitor of HIV would not be expected to possess a specific inhibitory effect on a toxic material such as Vpr, and *vice versa*.

In view of the common knowledge in the relevant art, as explained above, the skilled artisan would have differentiated “antiviral activity” from the “anti-toxin activity,” and would not have recognized “an antiviral treatment” to encompass “the inhibition of the activity of a toxic material/substance,” as presently recited.

It is axiomatic that an anticipating reference must describe each element of the claimed invention. As applicants have noted, however, Matthews *et al.* does not teach each and every element of the claimed method of inhibiting the activity of a toxic material/substance, and thus does not qualify as an anticipation of claims 1-12. Accordingly, reconsideration and withdrawal of the subject rejection are requested.

IV. Double Patenting Rejection

The examiner has rejected claims 1, 8 and 13 as claiming the same inventions as those of claims 1, 6 and 37 of U.S. Patent No. 6,190, 650 ("the '650 patent"). The examiner further has rejected claims 1-13 in view of claims 1-38 of the '650 patent under the judiciary created doctrine of obviousness-type double patenting. Applicants respectfully traverse this rejection.

While canceling claim 13, applicants would emphasize at the outset that, because claims 1 and 8 are directed to a method of inhibiting the activity of a toxic material/substance, they do not recite the same subject matter as do claims 1, 6 and 37 of the '650 patent, which are directed to a composition. The difference of these inventions evident from the examiner's own statement, made in rejecting claims 1-13 under obviousness-type double patenting. In the office action at page 5, section 7, the examiner admits that "the conflicting claims are not identical." For this very reason, applicants submit that the double patenting rejection of claims 1 and 8 should be withdrawn.

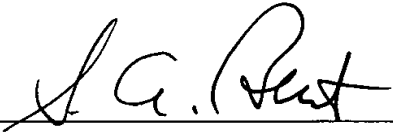
With respect to rejection under obviousness-type double patenting, applicants respectfully submit that the examiner's rationale for rejection should not stand in light of the distinctions that pertain in this context as elaborated above. In particular, applicants would emphasize that claim 1 is directed to a method of inhibiting the activity of a toxic material/substance, not to a method for antiviral treatment. For the reasons developed previously the skilled artisan would have differentiated an inhibition of toxic effects from an antiviral treatment. Therefore, claims 1-12 are patentably distinct from claims 1-38 of the '650 patent. Accordingly, reconsideration and withdrawal of all the double-patenting rejections are respectfully requested.

In view of the foregoing amendment and remarks, applicants respectfully request favorable reconsideration and allowance of the pending claims. If there are any issues remaining which the examiner believes could be resolved through either a

Supplemental Response or an Examiner's Amendment, the examiner hereby respectfully invited to contact the undersigned at the telephone number listed below.

Respectfully submitted,

Date 3 September 2002

By 

FOLEY & LARDNER
Customer Number: 22428



22428

PATENT TRADEMARK OFFICE

Telephone: (202) 672-5404

Facsimile: (202) 672-5399

Stephen A. Bent
Attorney for Applicant
Registration No. 29,768

VERSION WITH MARKINGS TO SHOW CHANGES MADE**Marked up replacement paragraph:**

Please replace the paragraph beginning on page 21 at line 14 with the following rewritten paragraph:

Preparation of sodium N-(2-sulfoethyl) succinamide terminated polylysine dendrimers

(SEQ ID NO: 3) BHAl₂lys₂lys₄lys₈[lys₆]lys₁₆, **BRI 2789**

[Trifluoroacetic] Trifluoroacetic acid (1ml) was added to a suspension of (SEQ ID NO: 2) BHAl₂lys₂lys₄lys₈[DBL₆]DBL₁₆ (36.5mg; 5.0μmol) in dry dichloromethane (1ml) and the resulting solution stirred at room temperature under nitrogen for two hours and then concentrated. The residue was dissolved in dry DMSO (2ml) and the pH adjusted to 8.5 with triethylamine. A solution of the crude tetrabutylammonium 4-nitrophenyl N-(2-sulfoethyl)succinamate (ca. 0.2mmol) in DMSO (1ml) was then added dropwise and the mixture stirred overnight at room temperature. The yellow solution was then concentrated (50 /10⁻⁵ mmHg) and the yellow residue partitioned between water and chloroform. The aqueous layer was separated, washed with chloroform (3X) and ethyl acetate, and then concentrated to give an oil (99mg). The crude product was converted to the sodium salt by passage through a column of Amberlite IR 120(Na) to yield 81 mg of material. This material was further purified by gel filtration ([Sephadex] Sephadex LH20; water) to give the sodium N-(2-sulfoethyl)succinamide terminated (SEQ ID NO: 3) BHAl₂lys₂lys₄lys₈[lys₆]lys₁₆ dendrimer (39mg). ¹³C nmr(D₂O):δ 27.0, 32.3, 35.2, 35.3, 35.6, 35.7, 39.5, 43.5, 54.1, 58.5, 131.5, 132.0, 133.3, 145.1, 177.8, 178.0, 178.4, 178.8, 178.9, 179.2, 179.7, 179.8.

Please replace the paragraphs beginning on page 22 at lines 4, with the following rewritten paragraph:

The corresponding [(SEQ ID NO: 1)] BHAl₂lys₂, (SEQ ID NO: 1) BHAl₂lys₂lys₄ (**BRI2787**) and (SEQ ID NO: 2) BHAl₂lys₂lys₄lys₈ .(**BRI2788**) terminated with sodium N-(2-

sulfoethyl)[succinamind] succinamide groups were similarly prepared. ¹³C nmr (SEQ ID NO: 2) BHAlyslys₂lys₄lys₈ derivative (D2O):δ 26.9, 32.3, 35.1, 35.3, 35.6, 35.7, 39.5, 43.5, 54.1, 58.5, 131.6, 131.9, 132.2, 132.3, 133.2, 133.3, 145.0, 145.2, 177.2, 177.8, 177.9, 178.0, 178.2, 178.3, 178.6, 178.7, 178.8, 178.9, 179.2, 179.3, 179.7, 179.8.

¹³C nmr (SEQ ID NO: 1) BHAlyslys₂lys₄ derivative (D2O):δ 26.9, 32.3, 35.1, 35.4, 35.7, 35.8, 39.5, 43.5, 54.1, 58.5, 61.8, 131.7, 132.0, 132.2, 132.3, 133.2, 133.3, 145.0, 145.1, 177.3, 178.0, 178.3, 178.4, 178.7, 178.9, 179.0, 179.3, 179.7, 179.8.

¹³C nmr BHAlyslys₂ derivative (D2O):δ 26.9, 27.1, 32.2, 32.3, 34.7, 34.8, 35.1, 35.3, 35.6, 35.7, 39.5, 43.4, 54.1, 58.6, 61.8, 131.7, 131.9, 132.2, 132.3, 133.3, 144.9, 145.0, 177.7, 178.4, 178.8, 179.0, 179.3, 180.0.

Please replace paragraph beginning on page 23 at line 19, with the following rewritten paragraph:

(SEQ ID NO: 3) BHAlyslys₂lys₄lys₈[lys₆]lys₁₆ **BRI2792**

Trifluoroacetic acid (4ml) was added to a suspension of (SEQ ID NO: 2) BHAlyslys₂lys₄lys₈[DBL₆]DB₁₆ (0.73g; 0.1mmol) in dry dichloromethane (4ml) under nitrogen. A vigorous evolution of gas was observed for a short time and the resulting solution was stirred at room temperature for two hours and then concentrated. The residual syrup was dissolved in water (5ml), the solution passed through a column of Amberlite IRA-401(OH) and the filtrate concentrated to give (SEQ ID NO: 3) BHAlyslys₂lys₄lys₈[lys₆]lys₁₆ as a viscous oil (0.49g). The oil was redissolved in water (5ml) and N,N-dimethyl-N-allylamine buffer (pH 9.5; 3ml) added. Solid sodium 4-sulfophenylisothiocyanate monohydrate (1.30g; 5.1mmol) was then added and the resulting solution heated under nitrogen at 53 °C for two hours and then cooled. The solution was concentrated and the brownish solid residue purified by gel filtration (Sephadex LH20; water). The pure fractions were combined, passed through a column of Amberlite IR 120(Na) and freeze dried to give the sodium 4-sulfophenylthiourea terminated (SEQ ID NO: 3) BHAlyslys₂lys₄lys₈[lys₆]lys₁₆ dendrimer as a fluffy white solid (374mg). ¹H nmr (D2O):δ 1.40; 1.72; 3.08; 3.42; 4.24; 4.60; 7.30; 7.40 (d, J=9Hz); 7.78 (d, J=9Hz). ¹³C nmr . (D2O):δ 27.3; 32.5; 35.9; 43.7; 48.9; 58.6; 63.3; 128.8; 131.0; 143.7; 144.7; 145.1; 177.7; 178.1; 183.8; 185.2.

Please replace the paragraph beginning on page 24 at line 8, with the following rewritten paragraph:

The corresponding (SEQ ID NO: 2) BHAlyslys₂lys₄lys₈[lys], (SEQ ID NO: 4) BHAlyslys₂lys₄lys₈lys₁₆lys₃₂ (**BRI2992**), and (SEQ ID NO: 5) BHAlyslys₂lys₄lys₈lys₁₆lys₃₂lys₆₄ (**BRI2993**) dendrimers terminated with 16, 64, and 128 sodium 4-sulfophenylthiourea groups respectively were similarly prepared.

Please replace the paragraph beginning on page 25 at line 13, with the following rewritten paragraph:

(SEQ ID NO: 3) BHAlyslys₂lys₄lys₈[lys₆]lys₁₆ **BRI2999**

Trifluoroacetic acid (2ml) was added to a suspension of (SEQ ID NO: 2) BHAlyslys₂lys₄lys₈ [DBL₆]DBL₁₆ (0.73g; 0.1mmol) in dry dichloromethane (2ml) under nitrogen. A vigorous evolution of gas was observed for a short time and the resulting solution was stirred at room temperature for two hours and then concentrated. The residual syrup was dissolved in water (5ml), the solution passed through a column of Amberlite IRA-401(OH) and the filtrate concentrated to give (SEQ ID NO: 3) BHAlyslys₂lys₄lys₈[lys₆]lys₁₆ as a viscous oil (0.49g). The oil was redissolved in water (5ml) and N,N-dimethyl-N-allylamine buffer (pH 9.5; 3ml) added. Solid sodium 3,6-sulfophenylisothiocyanate (234mg; 0.60mmol) was then added and the resulting solution heated under nitrogen at 53 °C for two hours and then cooled. The solution was concentrated and the brownish solid residue purified by gel filtration (Sephadex LH20; water). The pure fractions were combined, passed through a column of Amberlite IR 120(Na) and freeze dried to give (SEQ ID NO: 3) BHAlyslys₂lys₄lys₈[lys₆]lys₁₆ terminated with 32 sodium 3,6-disulfonaphthylthiourea groups as a fluffly off-white solid (119mg). ¹H nmr (D₂O):δ 1.0-2.0; 3.18; 3.43; 4.31; 7.22; 7.80; 7.89; 8.25. ¹³C nmr (D₂O):δ 27.2; 32.4; 35.3; 43.7; 49.0; 58.5; 63.6; 128.4; 129.1; 131.4; 136.1; 136.6; 138.6; 139.0; 145.6; 178.4; 184.8; 186.7.

Please replace the paragraph beginning on page 27 at line 18, with the following rewritten paragraph:

The corresponding sodium 3,6,8-trisulfonaphthylthiourea terminated dendrimer (SEQ ID NO: 3) BHAllyslys₂lys₄lys₈ [lys₆]lys₁₆ **BRI 7011** was prepared similarly. [The sweet potato sporamin vacuole]

Please replace the paragraph beginning on page 30 at line 9, with the following rewritten paragraph:

(SEQ ID NO: 3) BHAllyslys₂lys₄lys₈ [lys₆]lys₁₆ **BRI 2922**

Trifluoroacetic acid (4ml) was added to a suspension of (SEQ ID NO: 2) BHAllyslys₂lys₄lys₈ [DBL₆]DBL₁₆ (220mg; 30μmol) in dry dichloromethane (2ml) and the resulting solution stirred at room temperature under nitrogen for two hours and then concentrated. The residue was dissolved in dry DMSO (5ml) and the pH adjusted to 8.5 with triethylamine. Solid 4-nitrophenyl N,N,N-trimethylglycinate chloride (0.50g; 1.8mmol) was then added and the mixture stirred overnight at room temperature. The cloudy solution was then concentrated (50 /10⁻⁵ mmHg) and the residue partitioned between water and dichloromethane. The aqueous layer was separated, washed with dichloromethane (3X) and ethyl acetate, and then concentrated to give an oil (1.128g). The crude product was purified by gel filtration (Sephadex LH20; water) to give the N,N,N-trimethylglycinamide terminated (SEQ ID NO: 3) BHAllyslys₂lys₄lys₈ [lys₆]lys₁₆ dendrimer (116mg). ¹³C nmr (D₂O):δ 25.5, 30.5, 30.8, 33.4, 42.1, 56.5, 57.1, 67.5, 68.1, 166.7, 167.0, 167.1, 176.0, 176.2.

Please replace the paragraph beginning on page 39 at line 31 with the following rewritten paragraph:

(SEQ ID NO: 3) BHAlyslys₂lys₄lys₈[lys₆]lys₁₆ [8-(ocatnamido) octanamido]- 5-acetamido-3,5-dideoxy-2-thio D-glycero- α -D-galacto-2-nonulopyranosidoic acid]₃₂ **BRI 6169**

Please replace the paragraph beginning on page 40 at line 3 with the following rewritten paragraph:

A solution of (SEQ ID NO: 3) BHA lyslys₂lys₄lys₈lys₁₆ (t-Boc)₃₂ (20.3mg.) in a mixture of trifluoroacetic acid (2ml.) and dichloromethane (2ml.) was stirred at 20 C for 2 hours then solvent was removed under vacuum. The residue was dissolved in dry dimethyl sulphoxide (1ml.) and [id] di-isopropylethylamine (25mg.) and methyl [(8-octanoic acid N-hydroxysuccinimide ester) 5-acetamido-4,7,8,9-tetra-O-acetyl-e,5-dideoxy-2-thio-D-glycero- α -D-galacto-2-nonulopyranosid]onate (78mg.) were added. The mixture was stirred under argon at 20 C for 60 hours then solvent was removed under vacuum. The residue was dissolved in a freshly prepared 0.1M solution of sodium methoxide in methanol (2.5ml.) and the mixture stirred for 3 hours under argon at 20 C. The solvent was evaporated and the residue dissolved in water (1ml.) and stirred for 17 hours. This solution was subjected to size exclusion chromatography on Sephadex LH20 eluting with water. After lyophilisation, the product, (SEQ ID NO: 3) BHA lyslys₂lys₄lys₈lys₁₆ [(8-octanamido)-5- acetamido-3,5-dideoxy-2-thio-D-glycero- α - D-galacto-2-nonulopyranosidoic acid]₃₂ was obtained as a white powder 44mg. 86%.

Please replace the paragraph on page 13, between lines 21-24 with the following rewritten paragraphs:

[In the accompanying drawings:

Figures 1 to 4 show the effects of various concentrations of BRI 2923 in inhibition of the HIV toxic Vpr peptide fraction P3.]

Figure 1 Effect of 10e-4M BRI12923 on the cell toxicity caused by 10e-5M HIV Vpr peptide fraction P3 (not corrected).

Figure 2 Effect of 10e-4M BR12923 on the cell toxicity caused by 10e-5M HIV Vpr peptide fraction P3 (corrected).

Figure 1 Effect of 10e-4M BR12923 on the cell toxicity caused by 10e-5M HIV Vpr peptide fraction P3 (not corrected).

Figure 2 Effect of 10e-4M BR12923 on the cell toxicity caused by 10e-5M HIV Vpr peptide fraction P3 (corrected).

Figure 3 Effect of 10e-5M BR12923 on the cell toxicity caused by 2x10e-5M HIV Vpr peptide fraction P3 (not corrected).

Figure 4 Effect of 10e-5M & 10e-7M BR12923 on the cell toxicity caused by 2x10e-5M HIV Vpr peptide fraction P3 (not corrected).

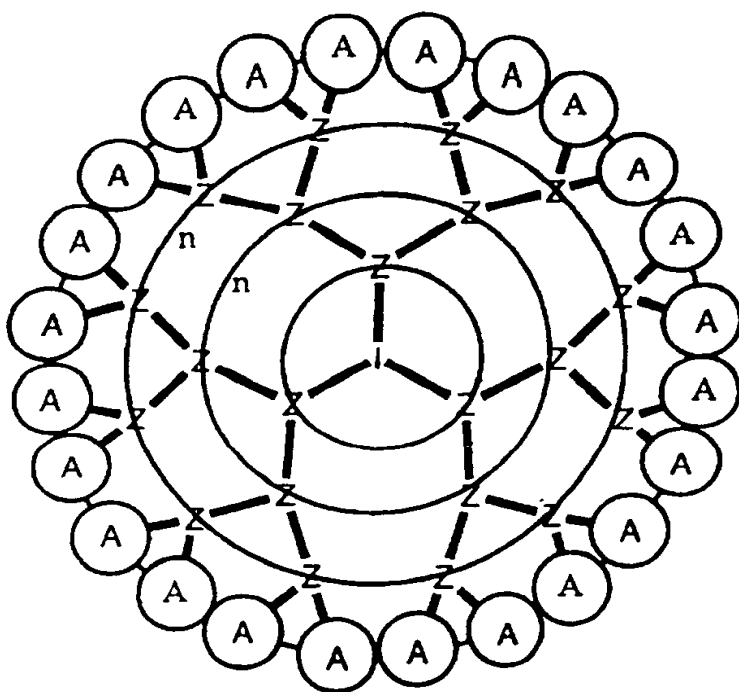
Marked up rewritten claims

1. (Amended) A method of [prophylactic or therapeutic inhibition of] inhibiting the activity of a toxic material or substance in a human or non-human animal patient, which comprises administration of the patient of an effective amount of a dendrimer having a plurality of terminal groups wherein at least one of said terminal groups has an anionic- or cationic-containing moiety bonded or linked thereto.

2. (Amended) A method according to claim 1, wherein said [compound is a] dendrimer [which] comprises a polyvalent core covalently bonded to at least two dendritic branches, and extends through at least two generations.

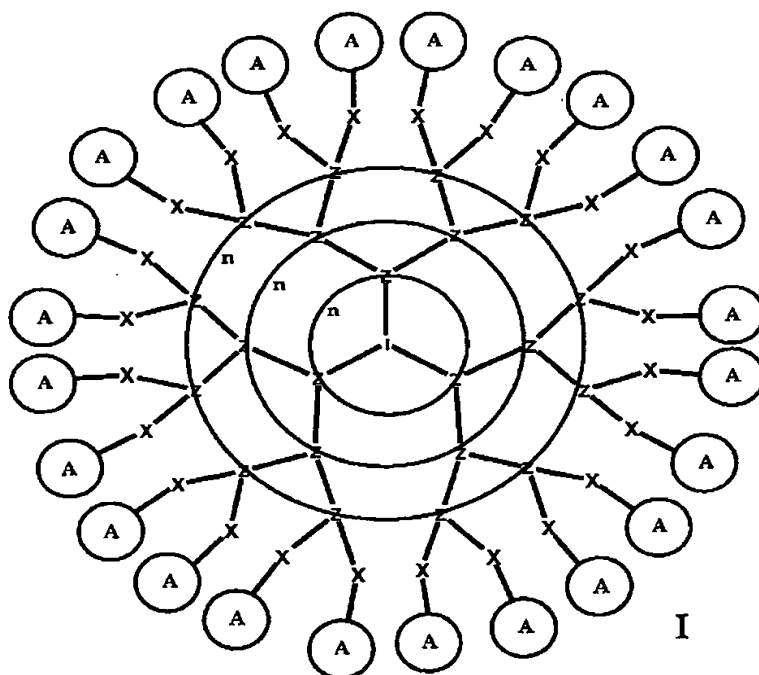
7. (Amended) A method according to claim 2 wherein said [compound] dendrimer is a polyionic dendrimer of the general formula I:

I



I

I



I

wherein:

I is an initiator core;

Z is an interior branching unit;

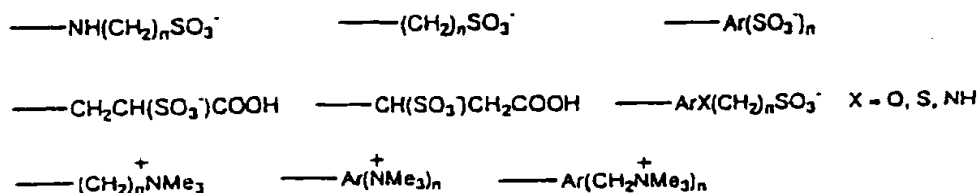
n is an integer which represents the number of generations of the dendrimer; and

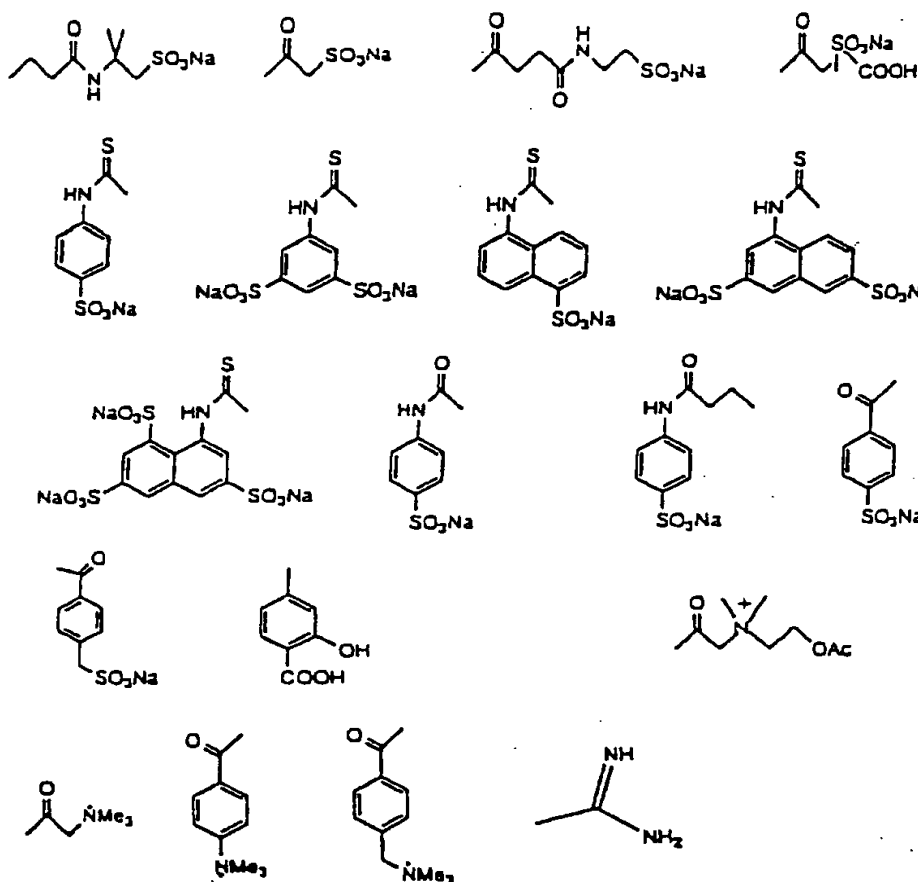
A is an anionic- or cationic containing moiety which may be linked to interior branching unit Z through an optional linking group X.

8. (Twice Amended) A method according to claim 1, wherein in said [compound] dendrimer said anionic- or cationic-containing moiety or moieties are bonded to amine, sulfhydryl, hydroxy or other reactive terminal groups of the dendrimer by amide or thiourea linkages.

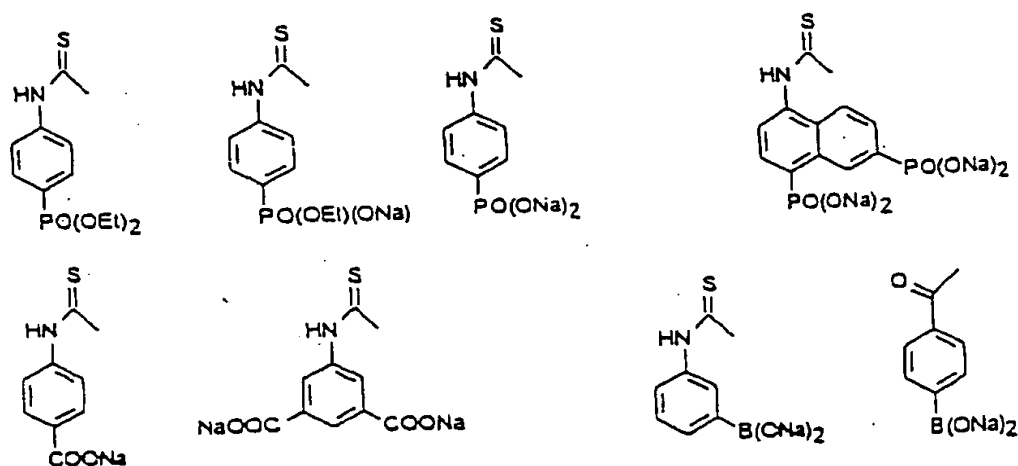
9. (Twice Amended) A method according to claim 1, wherein in said [compound] dendrimer said anionic- or cationic-containing moieties are selected from the group consisting of sulfonic acid-containing moieties, carboxylic acid-containing moieties, [(including] neuraminic and sialic acid-containing moieties, [and] modified neuraminic and sialic acid-containing moieties[)], boronic acid-containing moieties, phosphoric and phosphonic acid-containing moieties, [(including] esterified phosphoric and phosphonic acid-containing moieties[)], primary, secondary, tertiary or quaternary amino-containing moieties, pyridinium-containing moieties, guanidinium-containing moieties, amidinium-containing moieties, phenol-containing moieties, heterocycles possessing acidic or basic hydrogens, and zwitterionic-containing moieties.

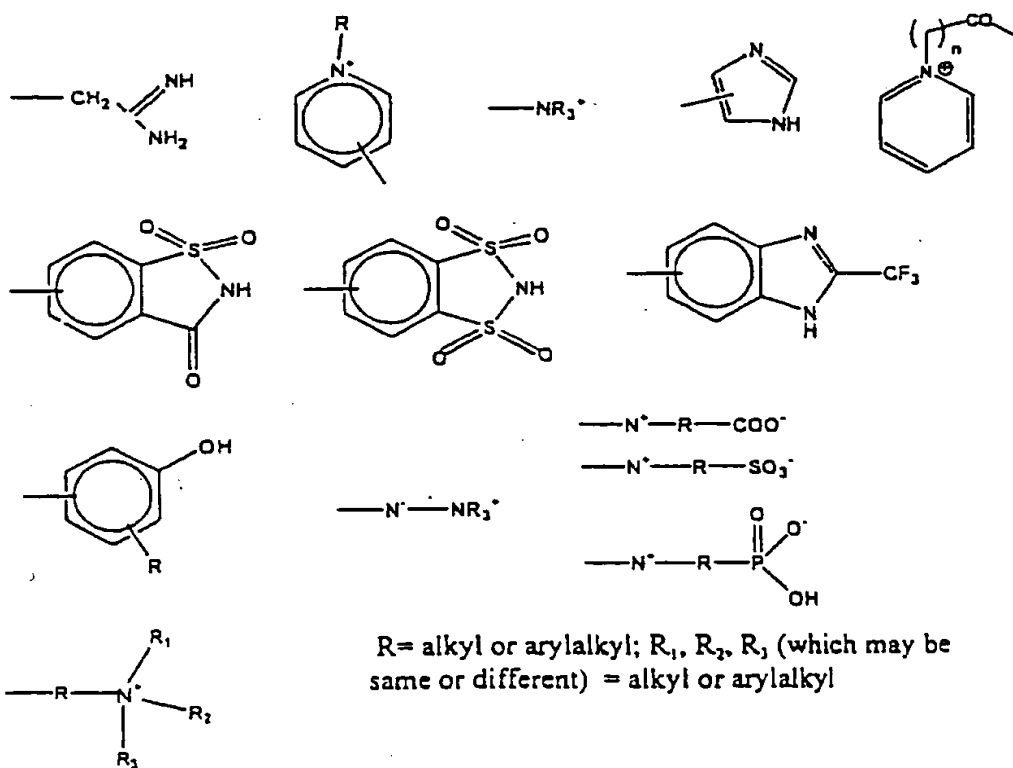
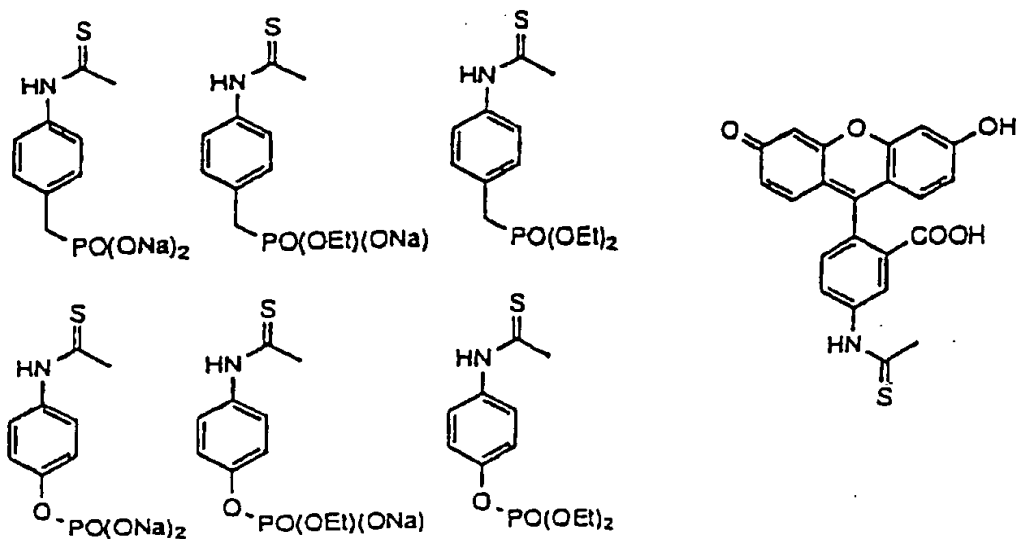
10. (Twice Amended) A method according to claim 1, wherein in said [compound] dendrimer the moiety or moieties which are bonded to amino or other reactive terminal groups of the dendrimer are selected from the following groups, in which n is zero or a positive integer:




$$\text{---ArXP(=O)(OR)}_2 \quad \text{X=O, CH}_2, \text{CHF, CF}_2 \quad \text{R=alkyl, aryl, H, Na.}$$

—ArXP(=O)(OR¹)(NR²R³) X=O, CH₂, CHF, CF₂ R¹=alkyl, aryl, H, Na R², R³=alkyl, aryl

$$\text{---Ar[P(=O)(OR)}_2\text{]}_n$$
 R=alkyl, aryl, H, Na n=1-3
$$\text{---Ar[B(OH)}_2\text{]}_n \quad n=1-3 \qquad \text{---Ar[COOH]}_n \quad n=1-3$$




11. (Twice Amended) A method according to claim 1, wherein said [compound] dendrimer is selected from the group consisting of:

- xli. alkylsulfonic acid terminated dendrimers;
- xlvi. sulfoacetamide terminated dendrimers;
- xlvi. sulfosuccinamic acid terminated dendrimers;
- xliv. N-(2-sulfoethyl) succinamide terminated dendrimers;
- xliv. 4-sulfophenylthiourea terminated dendrimers;
- xlvi. 3,6-di-sulfonaphthylthiourea terminated dendrimers;
- xlvi. 4-sulfonaphthylthiourea terminated dendrimers;
- xlvi. 3,5-di-sulfophenylthiourea terminated dendrimers;
- xlvi. 3,6,8-tri-sulfonaphthylthiourea terminated dendrimers;
- l. 4-(sulfomethyl) benzamide terminated dendrimers;
- li. 4-sulfobenzamide terminated dendrimers;
- lii. N-(4-sulfophenyl) propanamide terminated dendrimers;
- liii. 4-sulfophenylurea terminated dendrimers;
- liv. N,N,N-tri-methylglycinamide terminated dendrimers;
- lv. 4-trimethylammonium benzamide terminated dendrimers;
- lvi. 4-(trimethylammoniummethyl)benzamide terminated dendrimers;
- lvii. N-(2-acetoxyethyl)-N,N-(dimethylammonium)methyl-carboxamide terminated dendrimers;
- lviii. guanidino terminated dendrimers;
- lix. 4-([1,4,8,11-tetraazacyclotetradecane]methyl)benzamide terminated dendrimers;
- lx. 4-carboxy-3-hydroxy-benzylamine terminated dendrimers;
- lxi. 4-carboxyphenylamide terminated dendrimers;
- lxii. 3,5-dicarboxyphenylamide terminated dendrimers;
- lxiii. 4-phosphonooxyphenylthiourea terminated dendrimers;
- lxiv. 4-(phosphonomethyl)phenylthiourea terminated dendrimers;
- lxv. ethyl-4-(phosphonomethyl)phenylthiourea terminated dendrimers;
- lxvi. (8-octanamido)-5-acetamido-3,5-dideoxy-2-thio-D-glycero-D-galacto-2-nonulopyranosidoic acid terminated dendrimers;

- lxvii. (11-undecanamido)-5-acetamido-3,5-dideoxy-2-thio-D-glycero- β -D-galacto-2-nonulopyranosidoic acid terminated dendrimers;
- lxviii. (acetamido)-5-acetamido-3,5-dideoxy-2-thio-D-glycero- β -D-galacto-2-nonulopyranosidoic acid terminated dendrimers;
- lxix. (4-butanamido)-5-acetamido-3,5-dideoxy-2-thio-D-glycero- β -D-galacto-2-nonulopyranosidoic acid terminated dendrimers;
- lxx. (4-methylbenzamido)-5-acetamido-3,5-dideoxy-2-thio-D-glycero- β -D-galacto-2-nonulopyranosidoic acid terminated dendrimers;
- lxxi. (8-octanamido)-4-azido-5-acetamido-3,4,5-trideoxy-2-thio-D-glycero- β -D-galacto-2-nonulopyranosidoic acid terminated dendrimers;
- lxxii. (8-octanamido)-4-amino-5-acetamido-3,4,5-trideoxy-2-thio-D-glycero- β -D-galacto-2-nonulopyranosidoic acid terminated dendrimers;
- lxxiii. 4-benzamidoboronic acid terminated dendrimers;
- lxxiv. 3,5-dicarboxyphenylthiourea terminated dendrimers;
- lxxv. 4-phosphonooxyphenylthiourea terminated dendrimers;
- lxxvi. 4-phosphonophenylthiourea terminated dendrimers;
- lxxvii. 4,6-diphosphononaphthylthiourea terminated dendrimers;
- lxxviii. fluoresceinthiourea terminated dendrimers;
- lxxix. (phenyl-3-boronic acid)-thiourea terminated dendrimers;
- lxxx. pyridinium dodecylcarboxamide terminated dendrimers; and
saccharin terminated dendrimers.

12. (Twice Amended) A method according to claim 1, wherein [said treatment] said method comprises inhibition of toxins and toxic peptides of biological origin or toxins and toxic peptides released during bacterial, protozoal, fungal or viral infection.